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IPEX Journal

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Contents

Editorial

Bridging the Gaps in Practice: Toward a Unified Rehabilitation Protocol for Musculoskeletal Disorders

Umma S, Rahman A

Rehabilitation is more than a clinical process, it is the essence of regaining dignity, independence, and a sense of purpose in life. Rehabilitation helps an individual to achieve the highest possible level of function and quality of life-physically, emotionally, socially and vocationally after illness, injury or disability. According to the Global Burden of Disease Study 2019, approximately 2.4 billion individuals across the globe are in need of rehabilitation services[1]. Among this large population, a significant portion is concentrated in low- and middle-income countries (LMICs), where health systems are often under-resourced, under-prioritized, and under-researched. Like many other LMICs, the landscape of rehabilitation services for persons with disabilities (PwDs) in Bangladesh remains largely undocumented, inadequately structured, and inconsistently delivered. Therefore, the urgency of reform is undeniable.

Bangladesh presents a sobering picture: there are only 6.8 rehabilitation units per 1 million people, and only 6.2% of these facilities are situated in remote areas. Furthermore, the availability of skilled rehabilitation professionals is alarmingly insufficient. The current statistics indicate that there are merely 9.4 physiotherapists per 1 million people[2]. This scarcity is not just a number; it translates into prolonged suffering, untreated impairments, increased dependency, and reduced quality of life for millions.

To understand the magnitude of the issue, it is imperative to revisit what rehabilitation truly means and why it holds a central place in human recovery and dignity. Rehabilitation is defined as "a set of interventions designed to optimize functioning and reduce disability in individuals with health conditions in interaction with their environment" (World Health Organization. Rehabilitation. 2023). Rehabilitation is not merely a treatment modality; it is a lifeline. It is the bridge between injury and independence, illness and autonomy, trauma and transformation. Imagine a young man who, after a road traffic accident, is left sided paralyzed. Without rehabilitation, he is confined to a wheelchair, dependent on others for the simplest tasks. But through structured, consistent rehabilitation, he may regain enough strength and coordination to wheel himself outdoors, feel the breeze again, enjoy a morning walk with assistance, or even dream of returning to work. Rehabilitation gives people the opportunity to reclaim their lives, their roles in society, and their sense of self-worth. It allows individuals to once again to bathe in the rain, enjoy the twilight, walk under the moonlight, or simply rise from bed with dignity.

The concept of evidence-based rehabilitation protocol is essential in ensuring that such transformative care is standardized, effective, and widely accessible. Evidence-based rehabilitation refers to the integration of the best available clinical evidence from systematic research with the clinical expertise of professionals and the values and preferences of the patient[3]. This model provides a framework for consistent, efficient, and patient-centered care. The success of such approaches has been widely documented in various high-income countries. For example, in the United States, the American Physical Therapy Association (APTA) has developed comprehensive Clinical Practice Guidelines (CPGs) for various musculoskeletal conditions[4]. These guidelines are systematicallydeveloped statements to assist practitioner and patient decisions about appropriate healthcare for specific clinical circumstances. Similarly, the North American Spine Society (NASS) has published evidence-based clinical guidelines for the diagnosis and treatment of spine-related disorders, relying on rigorous literature reviews and expert consensus[5]. The American Academy of Orthopaedic Surgeons (AAOS) follows a similar model, incorporating randomized controlled trials and systematic reviews to develop best-practice protocols for conditions like rotator cuff tears or anterior cruciate ligament (ACL) injuries[6]. The Cochrane Rehabilitation Group takes this a step further by performing high-quality meta-analyses and systematic reviews to inform global rehabilitation practices[7]. Their recommendations are used to update

national policies and clinical training modules in several countries. These examples demonstrate how structured, evidence-informed approaches to rehabilitation can result in more equitable, effective, and sustainable care delivery. However, despite the clear benefits and international precedent, Bangladesh currently lacks a centralized rehabilitation society or authoritative body dedicated to developing such protocols. This absence has led to disjointed practices, inconsistent treatment outcomes, and regional disparities in the quality of care. Without a national framework or professional coalition to guide practice, rehabilitation services remain ad hoc and reactive rather than proactive and strategic.

Given these challenges, the need to develop a unified, evidence-based rehabilitation protocol in Bangladesh is not just urgent—it is crucial. This protocol should encompass a range of musculoskeletal conditions, be grounded in global best practices, and be adapted to local cultural, economic, and logistical realities. In this context, the importance of an integrated, evidence-based musculoskeletal rehabilitation protocol can not be overstated. The development of an integrated, evidence-based musculoskeletal rehabilitation protocol is essential for improving patient care in Bangladesh, where access to structured physiotherapy remains limited. In response, organizations such as the Institute for Professional Excellence & Research (IPEX), in collaboration with clinical partners including Micare Health & Research, have begun implementing protocols informed by clinical practice guidelines(CPGs), international protocols, high-quality randomized controlled trials and expert consensus. These efforts also involve the adoption of modern assessment tools such as the Muscle Meter, MAT assessment tool, Digital Lachmeter, Peripheral Neuropathy Scanner for comprehensive patient evaluation and outcome measurement. However, the reach of this initiative will remain limited without robust governmental support, especially to extend services to the remote and underserved areas of the country.

The current rehabilitation scenario in Bangladesh remains distressingly inadequate. In most cases, physiotherapy chambers are operated by medical technologists rather than qualified physiotherapists. Consequently, treatment strategies are often heavily dependent on electrotherapy modalities rather than on active, exercise-based rehabilitation. Moreover, patient education is largely absent. A striking example lies in the treatment of low back pain-while high-quality evidence strongly supports exercise-based rehabilitation for this condition, most clinics in Bangladesh continue to rely primarily on modality-based treatments.

An equally unfortunate reality is the lack of a functional referral system. Patients are rarely referred to physiotherapists after injuries, trauma, or surgeries, and in many cases, are not even informed about the importance of rehabilitation. This systemic failure can have life-altering consequences. In total knee arthroplasty (TKA) patients, studies have shown that muscular strength and power can be reduced by at least 24% in the operated limb compared to the sound limb. Researchers suggest that structured and progressive rehabilitation regimens are essential to restore strength and function post-surgery[8]. In 2010, for example, my grandfather developed left-sided hemiplegia as a result of an ischemic stroke. At the time, our family was living in a small town in Bangladesh. We had no awareness of physiotherapy, and no neurologist advised us to consult a physical therapist for rehabilitation. Consequently, my grandfather spent the rest of his life bedridden—an outcome that could likely have been prevented with timely and proper rehabilitation.

Rehabilitation services in Bangladesh are largely provided by the private sector. The government operates a limited number of rehabilitation centers, and these are largely situated outside the mainstream public health service delivery system. Rehabilitation is conspicuously absent from most of the primary and secondary health facilities, while the few services available at tertiary-level hospitals are often delivered by medical technologists. A growing concern is the emergence of non-medical individuals providing physiotherapy after attending short-term courses of six months or slightly more. This results in a severe shortage of trained personnel, an inequitable distribution of services, and high out-of-pocket costs. The cumulative effect is poor rehabilitation service uptake among persons with disabilities and chronic neglect of their health needs.

In conclusion, the creation of an integrated, evidence-based rehabilitation protocol for musculoskeletal disorders in Bangladesh represents not just a technical necessity but a moral imperative. To ensure its effectiveness and relevance, this protocol should be developed through a collaborative, interdisciplinary approach involving physiotherapists orthopedic surgeons, sports medicine specialists. These professional groups may work in synergy, combining their clinical expertise and practical experience to design a documented rehabilitation pathway that is both evidence-informed and patient-centered to bridging gaps between diagnosis, treatment, and long-term recovery, ultimately improving functional outcomes for patients. It is a call to action to bridge the gaps in our current healthcare delivery system, restore dignity to those living with disabilities, and align our national practices with international standards of care. It is time for Bangladesh to invest in the transformative potential of rehabilitation—not only to heal bodies, but also to rebuild lives, restore hope, and strengthen our national resilience.

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1. Umma Salma
Physiotherapist
Micare Health & Research
Email : ummasalma307@gmail.com

2. Dr. Abidur Rahman
Chief coordinator (Research)
Institute for Professional Excellence & Research
Email : abidavid@yahoo.com

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Original Article

The Role of Musculoskeletal Ultrasound in Enhancing Diagnostic Accuracy for Shoulder Disorders: Our experience in a specialized center

Rafi MAA[1], Mahdee MTA[2], Rahman A[3], Chowdhury MNR[4], Hossain KS[5]
Chowdhury MH[6], Islam S[7], Mamun A[8], Hossain MA[9]

Abstract

Background: Though shoulder pain is one of the most prevalent musculoskeletal symptoms, clinical diagnosis is sometimes difficult due to complex shoulder joint anatomy. Traditional diagnostic methods often fall short, hence advanced imaging techniques such as musculoskeletal ultrasonography is necessary.

Objective: Aim of the study is to assess the impact of MSKUS on diagnosis and management of shoulder pain by comparing pre-scan diagnosis and management plan with post scan assessment. This study also aims to emphasize the use of MSKUS for orthopedic shoulder surgeons.

Methods: This is a cross-sectional study conducted from January 2023 to January 2024. Patients aged 18 and older with acute or chronic shoulder pain were referred for ultrasound evaluation after initial diagnoses by primary clinicians. A total of 250 patients were enrolled. MSKUS examinations were performed by an experienced orthopedic surgeon who was blinded to the initial diagnoses. The changes in diagnosis and management plans pre- and post-ultrasound were statistically analyzed.

Results: Among the 250 patients, 76% had a change in diagnosis after MSKUS, with the most common pre-scan diagnosis being adhesive capsulitis (20.8%), while the post-scan diagnosis predominantly revealed rotator cuff tears (22.4%). Management plans also shifted significantly, with only 34.4% of patients advised conservative treatment post-scan compared to 92.4% pre-scan. Notably, 66% of patients experienced a change in management strategy.

Conclusion: The incorporation of MSKUS significantly alters the clinical diagnosis and management of shoulder pain, emphasizing its role as a valuable tool for orthopedic surgeons. This study advocates for increased utilization of MSKUS in routine clinical practice to enhance diagnostic accuracy and optimize treatment strategies for shoulder conditions.

Keywords: Shoulder pain, musculoskeletal ultrasonography (MSKUS), rotator cuff tears, adhesive capsulitis, imaging techniques, clinical diagnosis, shoulder joint.

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Introduction

Shoulder pain is the 3rd most common musculoskeletal symptom after knee and low back pain affecting 20.9–26% population globally[1]. The underlying causes of shoulder pain is diverse. Rotator cuff arthropathy, major and minor rotator disease, long head biceps and labral pathology, glenohumeral disorders, acromioclavicular joint (ACJ) pathology, and referred neck pain are the most common etiology[2]

Initial method for diagnosing shoulder pathology are clinical examinations and X-rays. However, these approaches often fall short of capturing the full complexity of shoulder disorders highlighting the need for high-resolution imaging techniques, particularly ultrasound and MRI.

1. Dr. Abdullah Al Rafi, Chief consultant & Coordinator, Micare Health & Research, Shyamoli, Dhaka, Bangladesh
2. Dr. Mujtaba Tamim Al Mahdee, Research Associate, Micare Health & Research, Shyamoli, Dhaka, Bangladesh
3. Dr. Abidur Rahman, Registrar (Surgery) Khulna City Medical College Hospital, Khulna
4. Dr. Muhammad Nasim Reza Chowdhury, Rheumatologist, Cumilla Medical College Hospital, Cumilla
5. Dr kazi Shahadat Hossain, Associate professor and Head, Dept of orthopaedics , Mainamoti medical college and Hospital, Cumilla
6. Dr.Monzur Hussain Chowdhury, Sports Physician Bangladesh Cricket Board Association- AMFSEM (UK)
7. Dr. Shohidul Islam, Resident,National Institute of traumatology and orthopedic rehabilitation
8. Dr Al Mamun, Assistant Regiastrar, Manikganj Medical college Hospital
9. Dr. Md. Amran Hossain, Consultant & Coordinator, Micare Physiotherapy and Rehab Center, Shyamoli, Dhaka, Bangladesh

Corresponding Author: Dr. Abdullah Al Rafi, Chief consultant & Coordinator, Micare Health & Research, Shyamoli, Dhaka, Bangladesh
Email: rafi123.sh3@gmail.com

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Among these options, Musculoskeletal ultrasound (MSKUS) has emerged as a crucial tool for assessing shoulder pathology. It provides real-time, dynamic, and functional imaging of soft tissues, tendons, and joints, allowing for detailed visualization of shoulder structures during movement. Additionally, MSKUS is more cost-effective than MRI, non-invasive, widely accessible, and has shown diagnostic accuracy comparable to that of MRI[3–5]. On the other hand, Complexity of shoulder anatomy is also a limitation in providing definitive diagnosis in clinical evaluation. As a result, Clinical diagnosis can be inaccurate or incomplete leading to inappropriate management plan.

A key advantage of MSKUS is that it enables an orthopedic surgeon to make bedside diagnosis and develop immediate, effective treatment plans. Surgeons have the benefit of a complete clinical history and shoulder examination, along with a better understanding of the patient's clinical issues. Also, Previous research has shown that shoulder surgeons, with adequate training, can effectively perform ultrasound examinations with similar effectiveness to radiologists[6]. Therefore, they can use ultrasound as an extension of their clinical assessment, increasing diagnostic accuracy and decision-making.

However, despite its advantages MSKUS remains underutilized among orthopedic surgeons[7,8]. particularly due to its operator-dependent nature and the limited ultrasound training

received by many practitioners. Successful implementation requires skill in probe positioning for optimal image acquisition and thorough understanding of shoulder anatomy in standardized planes[3].

This study aims to assess how the introduction of MSK USG impacts the clinical diagnosis and management of patients with shoulder pain. By comparing the initial clinical assessments made by primary physician with subsequent diagnoses obtained through ultrasound made by another, we seek to understand the extent to which MSK USG alters clinical impressions and treatment plans. The study's findings may emphasize the importance of integrating ultrasound into routine clinical practice of an orthopedic shoulder surgeon for the assessment and management of shoulder pain.

Materials & Methods:

Study design:

This cross-sectional study was conducted from January 2023 to January 2024 at a specialized center for minimally invasive spine and orthopedic surgery. Institutional ethics research board approval was obtained for this cross-sectional study.

Study population:

Our study population consisted of patients who were referred to our center for ultrasonographic evaluation with a prior diagnosis and management plan made by the index clinicians. Adult individuals of either sex, aged 18 years and older, with acute or chronic shoulder pain, who were referred to our center for sonographic evaluation and had provided written informed consent to participate in the study, were considered for enrollment. However, patients who had undergone recent surgery within the last 6 weeks, had acute shoulder injuries (e.g., fractures, dislocations) requiring immediate surgical intervention, or were unable to provide consent were excluded from the study. A total of 250 patients met the inclusion criteria, and 102 patients were excluded.

Evaluation method:

The referred patients were evaluated through ultrasound by the corresponding author of this study, an orthopedic surgeon with 10 years of experience in musculoskeletal ultrasonography, using a standardized and widely accepted protocol and technique (9). He was blinded to the diagnosis and management plan made by the index clinician. After performing MSKUS, the corresponding author then made his diagnosis and management plan according to sonographic findings. Both the pre- and post-ultrasound diagnoses, along with the management plans, were analyzed by other authors of this study, doctors and research associates of the study center. The treatment plans considered were, 1. conservative; 2. non-surgical intervention; 3. Surgery. Then the information was updated on the standard record sheet whether the diagnosis and management plan had changed or remained unchanged.

Imaging Method:

All ultrasound examinations were conducted using a LOGIQ P6 Pro scanner (GE Healthcare) equipped with a 6-15 MHz linear array transducer. The patient was seated comfortably in a chair, while the examiner either stood behind or sat beside the patient. Static and dynamic assessments of the shoulder joint were performed in both the transverse and longitudinal planes. Bilateral examination was also done when required.

Statistical analysis:

Data were analyzed for changes in diagnosis and management plan based on US results by nonparametric statistical methods using SPSS© version 27. 11

Results:

Baseline Characteristics:

A total of 250 referred patients were enrolled for this study. The majority of the patients were female (56.8%), aged ≥ 41 years (63.2%), with anterior shoulder pain (40.4%) and focal restriction of the joint movement (54.8%). A considerable proportion of the patients had diffuse shoulder pain (32.4%) and global restriction of movement (43.6%). The remaining demographic

characteristic and clinical findings are summarized in Table 1.

Baseline characteristics	N=250	P (%)
Age in years		
Mean ± SD (years)	48.63±13.82	
18-30 years	36	14.4
31-40 years	56	22.4
≥41 years	158	63.2
Sex		
Female	142	56.8
Chief Complaints		
Pain		
Diffuse	81	32.4
Anterior	101	40.4
Lateral	48	19.2
Posterior	16	6.4
Superior	4	1.6
Range of motion (ROM)		
Normal	4	1.6
Focal restriction	137	54.8
Global restriction	109	43.6

Pre-scan and post-scan diagnosis:

The study involved 250 patients initially diagnosed with various shoulder conditions. Majority (76%) of our patients' diagnosis status changed after their shoulder sonography. However, the diagnosis status of the remain ing (24%) patients remains unchanged even after sonography. (Figure 1)

Figure 1: Diagnosis status of overall patients after shoulder sonography

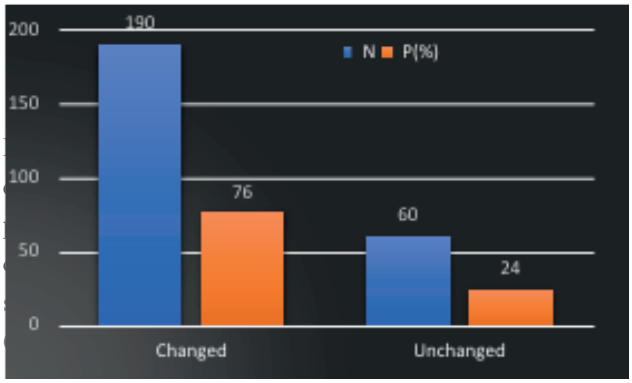


Table 2: Distribution of our study patients by pre-scan diagnosis

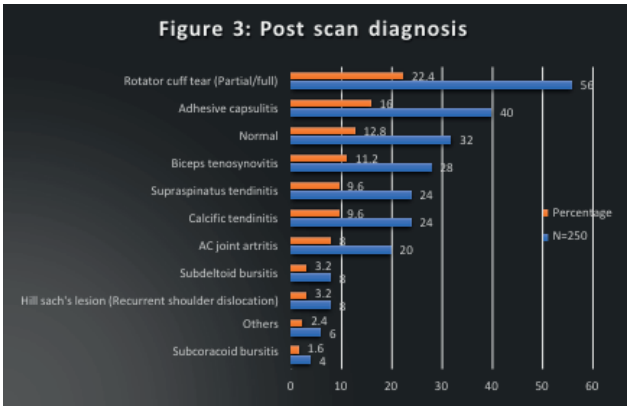
In pre-scan, the most frequent diagnosis was adhesive capsulitis, affecting 20.8% of patients, followed by post-traumatic right shoulder pain (14.4%), and rotator cuff tears (9.6%). Other common diagnoses included supraspinatus tendinitis (6.4%) and painful Arc syndrome (1.6%) (Table 2).

Table 2: Distribution of our study patients by pre-scan diagnosis

	N=250	P(%)
Pre-scan diagnosis		
Unspecified Shoulder pain	117	46.8
Adhesive Capsulitis	52	20.8
Traumatic Shoulder Pain	36	14.4
Rotator Cuff Tear (Partial/Full)	24	9.6
Supraspinatous Tendinitis	16	6.4
Painful Arc Syndrome	4	1.6
Hill Sach's Lesion	4	1.6
(Recurrent Shoulder Dislocation)		

After the scan, the diagnoses shifted significantly. The majority of the patients were found to have rotator cuff tear (Partial/full) (22.4%). Adhesive capsulitis which was most frequent in pre-scan, was found in 16% of the patients in post-scan. (Figure 2)

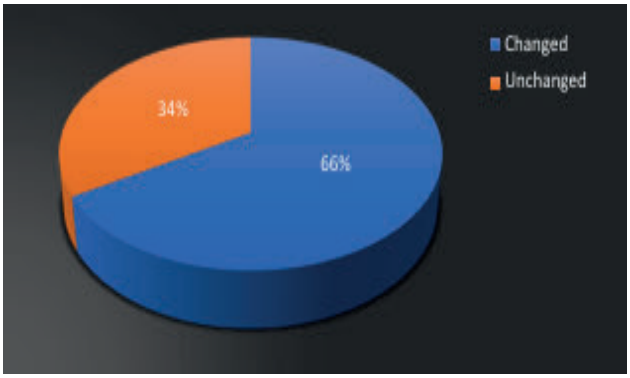
Figure 2: Distribution of our study patients by post-scan diagnosis



Pre-scan and post-scan management plan:

Majority (66%) of our patients' management plans changed after their shoulder sonography. However, the management plan of the remaining (34%) patients remains unchanged even after sonography. (Figure 3)

Figure 3: Management plan of overall patients after shoulder sonography



Before sonography, most of the patients (92.4%) were advised to go for the conservative treatment plan, and 7.6% for the non-surgical intervention. After shoulder sonography, the management plan of these patients changed. Only 34.4% of patients were advised for the conservative treatment plan, 59.6% of patients for the non-surgical intervention method, and 6% of patients were advised for surgery (Table 3).

Table 3: Comparison of management plan between before and after shoulder sonography

Management plan	Pre-scan		Post-scan	
	N	P (%)	N	P (%)
Conservative	231	92.4	86	34.4
Non-Surgical Intervention	19	7.6	149	59.6
Surgical			15	6
Total	250	100	250	100

Discussion:

This study demonstrates the impact of musculoskeletal ultrasonography in the diagnosis and management of shoulder pain. The clinical diagnosis of the majority of patients (76%) was changed after ultrasound scanning. Various studies have also shown that the post-scan diagnosis differs from the pre-scan clinical diagnosis in most shoulder pain patients[10].

Nearly 92.4% of the patients were initially treated conservatively. However, after the diagnostic shoulder US examination was performed, 65.6% of the patients were re-stratified to a more invasive treatment plan including a non-surgical intervention or surgery. These findings support our hypothesis that clinicians should emphasize integrating ultrasound into routine clinical practice for accurate assessment and proper management plan of shoulder pain. In the remaining 7.6% of the patients, the index clinician opted for non-surgical intervention, and no patient was given a surgical management plan. In both of these clinical settings, musculoskeletal shoulder US made a substantial impact. However, to our knowledge, there has been no investigation into the actual therapeutic impact of musculoskeletal shoulder US.

Clinically, most of the patients in our study had unspecified diagnoses. That is because common shoulder disorders often share similar clinical features, and the absence of consensus on diagnostic criteria, as well as inconsistencies in clinical assessments, complicates decisions.[11] Other than that, the most frequent pre-scan diagnosis was adhesive capsulitis, commonly known as "Frozen Shoulder".

Studies found the prevalence of adhesive capsulitis in the general population ranges from 2 to 5%. Generally, women and those over 40 years of age are at a greater risk.[12,13] In our study, rotator cuff tear, either full or partial was the most common diagnosis in post-scan. Several studies support this finding showing that rotator cuff tear is one of the most common causes of shoulder pain[14,15], most prevalent in middle-aged and older patients[16]. Considering the intricacy of shoulder disorders, the use of imaging techniques for diagnosis and management is essential. It is already established that the primary method for visualizing the soft tissues of the shoulder joint is ultrasound imaging[17]. Dynamic, real-time ultrasound imaging has proven effective in assessing both rotator cuff and non-rotator cuff shoulder conditions[18,19]. Also, the diagnostic accuracy of musculoskeletal ultrasound (MSK US) has been validated and shown to be comparable with MRI[20,21]. In an orthopedic surgeon's chamber, MSKUS can serve as a valuable tool for diagnostic accuracy. Additionally, it provides an opportunity to educate the patients and explain various management options[22].

In our study, the majority of the patients were female and aged ≥41 years. Studies support that the prevalence of shoulder pathology is associated with increasing age[23,24]. This may be attributed to the influence of physical activity and job-related factors on shoulder pain, a connection that has been established in other studies[24]. Also, studies showed that shoulder pain is more prevalent in women than in men.[25,26]

Limitations:

There were several limitations to our study. First, as all the patients were referred by different specialty doctors, pre-scan diagnoses were not made following a standard protocol. Therefore, the diagnosis and management plan might have been affected by the particular sequence and techniques used in their practices. This potential limitation could be addressed in a prospective trial including participating in treating clinicians as well as evaluating clinicians in pre- and post-US diagnosis in same setting. Finally, the assessment did not include prolonged patient follow-up for clinically recurring symptoms or responses to treatment. Another limitation of our study is the absence of a cost-effectiveness analysis. Future research should assess the economic benefits of using MSKUS compared to traditional diagnostic methods, such as X-rays or MRI, to better understand its value from both a clinical and financial perspective. This evaluation could provide further justification for the widespread adoption of MSKUS in clinical settings, ensuring that its benefits extend not only to patient outcomes but also to healthcare resource optimization.

Conclusion:

In conclusion, musculoskeletal shoulder ultrasound (MSKUS) is a well-established and validated diagnostic imaging modality for evaluating shoulder pain patients. In line with its recent increased utilization, this study demonstrates that musculoskeletal shoulder US significantly influences both patient treatment and clinician's decision-making processes. So, orthopedic shoulder surgeons should incorporate MSKUS in their routine clinical practice with proper training and skills.

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Declaration of competing interest:

None.

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Original Article

Estimating Acute Cholecystitis Severity Using C-Reactive Protein and ESR Levels

Sardar A[1], Rahman GS[2], Siddique MAB[3], Hossain MK[4], Galib A[5]

Abstract

Background: Acute cholecystitis (AC) is a common inflammatory condition of the gallbladder, and timely severity assessment is crucial for appropriate management. While the Tokyo Guidelines (TG18) provide a framework for diagnosis and grading, they lack specific biomarker thresholds for severity estimation. This study explores the role of C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) as potential predictors of disease severity.

Aim of the study: To evaluate the association of CRP and ESR levels with the severity of acute cholecystitis and propose potential cut-off values for clinical application.

Methods: A descriptive cross-sectional study was conducted on 130 patients diagnosed with acute cholecystitis between June 2023 and June 2025. Patients were classified into mild, moderate, or severe categories based on clinical, laboratory, and imaging findings. CRP and ESR levels were analyzed in relation to disease severity.

Result: CRP and ESR levels were significantly associated with disease severity ($p<0.001$). All mild cases had CRP <1 mg/L, while all severe cases had CRP >6 mg/L. Mean CRP values were 0.646±0.231 mg/L (mild), 5.30±1.76 mg/L (moderate), and 13.78±4.48 mg/L (severe). ESR followed a similar trend: 14.76±3.54 mm/hr (mild), 36.78±2.35 mm/hr (moderate), and 47.45±2.97 mm/hr (severe). Ultrasound findings such as gallbladder wall thickening, common bile duct dilation, and bile spillage were also significantly associated with severity.

Conclusion: CRP and ESR levels correlate strongly with the severity of acute cholecystitis and may serve as practical biomarkers for early risk stratification. These findings support their integration into routine diagnostic and prognostic evaluation, particularly in resource-limited settings.

Keywords: Acute cholecystitis, C-reactive protein, Erythrocyte sedimentation rate, Inflammation, Severity grading, Tokyo Guidelines.

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Introduction

Acute cholecystitis (AC) is one of the most common complications of gallstone disease and a leading cause of emergency hospital admissions for abdominal pain requiring surgical intervention[1]. Globally, gallstone disease affects approximately 10–20% of the adult population, with acute cholecystitis developing in up to 20% of symptomatic patients[2]. In Bangladesh, gallbladder disease has shown a rising trend, with hospital-based studies reporting a prevalence of 6–8% among adults, and acute cholecystitis accounting for a significant portion of gallstone-related admissions, especially among women aged 30–60 years[3]. Despite advances in diagnostic modalities and surgical

1. Dr. Anirudha Sardar, Resident Surgeon, Department of Surgery, Khulna Medical College Hospital, Khulna, Bangladesh
2. Dr. Gazi Shafiqur Rahman, Assistant Registrar, Department of Surgery, Khulna Medical College Hospital, Khulna, Bangladesh
3. Dr. Mohammad Abu Bakr Siddique, Assistant Professor, Department of Surgery, Khulna Specialist Hospital, Khulna, Bangladesh
4. Dr. Md. Kanak Hossain, Assistant Registrar, Department of Surgery, Khulna Medical College Hospital, Khulna, Bangladesh
5. Dr. Asadullahil Galib, Assistant Professor, Department of Surgery, Khulna Medical College Hospital, Khulna, Bangladesh

Corresponding Author: Dr. Anirudha Sardar, Resident Surgeon, Department of Surgery, Khulna Medical College Hospital, Khulna, Bangladesh, mail: dr.anirudha8@gmail.com, orcid id: 0000-0001-6092-6946

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techniques, early and accurate severity assessment of acute cholecystitis remains a key challenge for optimizing treatment decisions and improving patient outcomes[4]. The diagnosis and grading of acute cholecystitis were largely subjective and varied across institutions. The Tokyo Guidelines (TG), first proposed in 2007 and subsequently updated in 2013 and 2018, established internationally recognized diagnostic and severity grading criteria for acute cholecystitis to unify clinical practice and improve patient care[5]. These guidelines integrate clinical, laboratory, and radiological findings to improve diagnostic accuracy and guide therapeutic strategies[6]. According to the Tokyo Guidelines 2018 (TG18), the diagnosis of acute cholecystitis is based on a combination of local signs of inflammation e.g., Murphy's sign, right upper quadrant tenderness, systemic signs of inflammation e.g., fever, elevated white blood cell count, or elevated C-reactive protein [CRP], and imaging findings characteristic of gallbladder inflammation[7]. Following diagnosis, the clinical severity is stratified into three grades: mild, moderate, and severe, based on clinical, laboratory, and organ dysfunction parameters[8]. While CRP is listed in the TG18 as part of the diagnostic workup, it is not formally included in the severity grading criteria. However, emerging evidence suggests that CRP, as a sensitive and dynamic acute-phase reactant, correlates well with the severity of inflammation in acute cholecystitis and can serve as a useful prognostic indicator[9]. Numerous studies have demonstrated that higher CRP levels are associated with complicated or severe cases, such as gangrenous cholecystitis, empyema, or perforation[10]. Despite this, the guidelines do not propose specific CRP cut-off values for grading severity, leaving clinicians without a standardized threshold to differentiate mild from moderate or severe disease[11]. Similarly, erythrocyte sedimentation rate (ESR), although less frequently used in acute settings due to its slower kinetics, may complement CRP in identifying prolonged or subacute inflammation[12]. Given the wide availability, low cost, and clinical utility of CRP and ESR, establishing their predictive value for assessing the severity of acute cholecystitis may enhance early risk stratification, particularly in low-resource settings[13]. This study aims to evaluate the role of CRP and ESR in estimating disease severity among patients with acute cholecystitis, with the goal of determining potential cut-off values that can support clinical decision-making alongside the Tokyo Guidelines criteria.

Methodology & Materials

This descriptive cross-sectional study was conducted at the Department of Surgery, Khulna Medical College Hospital, Khulna, Bangladesh. The study was carried out over a period of two year, from June 2023 to June 2025. This study was designed as a diagnostic, observational cohort study aimed at evaluating the association between the severity of acute cholecystitis and levels of inflammatory markers, specifically CRP and ESR. The study was conducted in a clinical setting where all relevant laboratory and imaging assessments were performed as part of routine care. The study included total 130 patients with acute cholecystitis.

Inclusion Criteria

- Aged 18 years or older.
- Underwent serum CRP and ESR testing at the time of hospital admission.
- Diagnosed with acute cholecystitis based on clinical, laboratory, and ultrasonographic findings.

Exclusion Criteria

- Individuals with coexisting conditions known to influence inflammatory markers, such as diabetes mellitus, HIV, hepatitis, intestinal tuberculosis, or other immunocompromised states.
- Patients with histopathological findings suggestive of malignancy.
- Pregnant individuals.
- Patients with conditions that could confound CRP levels, such as acute pancreatitis or systemic infections.

Ethical Consideration

Approval was obtained from the Institutional Ethics Committee before the commencement of data collection. All participants were fully informed about the study's purpose, procedures, and any potential risks. Informed consent was obtained in written form. Confidentiality of patient information was maintained. As no experimental interventions were involved and only routine laboratory investigations were analyzed, the study posed minimal risk to participants.

Surgical Technique

Most patients underwent laparoscopic cholecystectomy under general anesthesia. The standard technique included the use of four ports (two 5 mm and two 10 mm) with visualization of Rouviere's sulcus and critical view of safety for identifying Calot's triangle. Cystic duct and artery were clipped, and the gallbladder was removed from the liver bed. In cases requiring open cholecystectomy, a right subcostal incision was made to facilitate gallbladder removal using electrocautery or harmonic scalpel. When indicated, cholangiography or common bile duct exploration was also performed.

Data collection

Medical records were reviewed to collect demographic data, including age, sex, and body mass index (BMI), as well as comorbidities. Laboratory investigations performed at initial presentation included complete blood count (hemoglobin and WBC), liver enzymes (ALT, AST), amylase, total bilirubin, CRP, and ESR levels. Radiological findings, including gallbladder wall thickness, common bile duct (CBD) diameter, number and size of gallstones, presence of impacted stones at the gallbladder neck, and bile spillage, were retrieved from ultrasound reports. The severity of acute cholecystitis was classified into mild (n=80), moderate (n=30), and severe (n=20) categories based on clinical, radiological, and surgical criteria.

Statistical Analysis

Data were entered and analyzed using SPSS version 26. Descriptive statistics were used to summarize the data. Categorical variables were presented as frequencies and percentages, while continuous variables were reported as mean ± standard deviation (SD). Associations between AC severity and categorical variables were analyzed using the Fisher's exact test or Chi-square test, as appropriate. A p-value less than 0.05 was considered statistically significant.

Result

The mean BMI was 25.7 ± 5.1 kg/m^2. Comorbidities were present in 24 patients (18.46%). The mean hemoglobin level was 12.5 ± 2.6 g/dL, and the average white blood cell count was 11.8 ± 3.7 ×10^3/mL. Liver function parameters showed a mean total bilirubin of 1.56 ± 1.28 mg/dL, ALT of 50.6 ± 51.4 IU/L, and AST of 55.3 ± 61.7 IU/L (Table 1). Female (55.38%) participants were more common than male (44.62%) participants (Figure 1). Mild cholecystitis was more common in younger age groups, particularly 31–40 years (37.5%) and 41–50 years (30%). Moderate and severe cases were more frequent in older patients, with 26.67% and 40% of moderate and severe cases, respectively, occurring in the 51–60 age group (Table 2). Table 3 shows that ultrasound findings showed significant associations with cholecystitis severity. Gallbladder wall thickening (>4 mm) was more prevalent in moderate (20%) and severe cases (30%) compared to none in mild cases (p=0.02). A dilated common bile duct (>6 mm) was observed in 26.67% of moderate and 40% of severe cases versus only 5% of mild cases (p=0.005). Multiple stones were more common in severe cholecystitis (50%) compared to mild (10%) (p=0.014). Larger stones (>1 cm) predominated in mild and moderate cases but were less frequent in severe cases (p=0.052). Impacted stones at the gallbladder neck were significantly associated with increasing severity (p=0.001), present in 50% of severe cases. Bile spillage was also strongly correlated with severity, occurring in 90% of severe and 33.33% of moderate cases but absent in all mild cases (p=0.001) (Table 3). All patients with mild cholecystitis had CRP levels below 1 mg/L. In moderate cases, 66.67% had CRP levels between 1–6 mg/L, while 33.33% had levels above 6 mg/L. All patients with severe cholecystitis had CRP levels greater than 6 mg/L. The mean CRP levels increased with severity: 0.646±0.231 mg/L in mild, 5.30±1.76 mg/L in moderate, and 13.78±4.48 mg/L in severe cases (Table 4). Among patients with mild cholecystitis, 87.5% had ESR levels between 14–18 mm/hour, and 12.5% had levels between 19–38 mm/hour. In moderate cases, 93.33% had ESR levels of 19–38 mm/hour, whereas 6.67% had levels of 39–50 mm/hour. All severe cases had ESR levels between 39–50 mm/hour. The mean ESR values increased with disease severity: 14.76±3.54 mm/hour in mild, 36.78±2.35 mm/hour in moderate, and 47.45±2.97 mm/hour in severe cholecystitis (Table 5).

Table 1: Baseline characteristics of study population (N=130)

Variables	Mean±SD
Comorbidity, n(%)	24 (18.46)
BMI (kg/m2)	25.7 ± 5.1
Hemoglobin (gr/dl)	12.5 ± 2.6
WBC x 103 mL	11.8 ± 3.7
Total bilirubin (mg/dl)	1.56 ± 1.28
ALT (IU/L)	50.6 ± 51.4
AST (IU/L)	55.3 ± 61.7

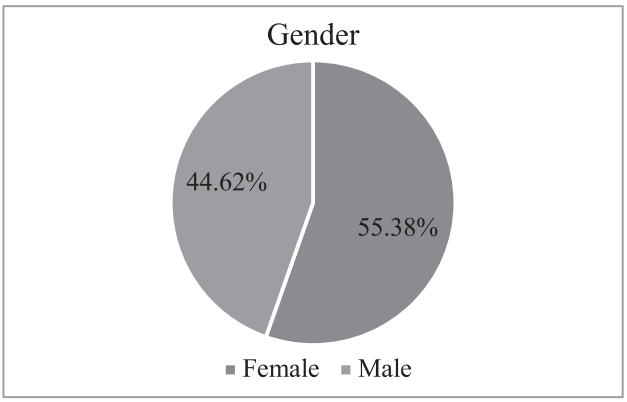

Figure 1: Distribution of patients by gender (N=130)

Table 2: Age distribution of participants (N=130)

Age (years)	Mild cholecystitis (n=80)		Moderate cholecystitis (n=30)		Severe cholecystitis (n=20)		P-value
	n	%	n	%	n	%	
<30	16	20.00	2	6.67	4	20.00	
31-40	30	37.50	2	6.67	2	10.00	
41-50	24	30.00	12	40.00	4	20.00	0.027
51-60	6	7.50	8	26.67	8	40.00	
>60	4	5.00	6	20.00	2	10.00	

Table 3: Association of grade of acute cholecystitis with USG findings (N=130)

USG	Mild cholecystitis (n=80)		Moderate cholecystitis (n=30)		Severe cholecystitis (n=20)		Total		P-value
	n	%	n	%	n	%	n	%	
Gall bladder wall thickness									
<4 mm	80	100	24	80	14	70	118	90.77	0.02
>4 mm	0	0	6	20	6	30	12	9.23	
CBD diameter									
<6 mm	76	95	22	73.33	12	60	110	84.62	0.005
>6 mm	4	5	8	26.67	8	40	20	15.38	
Number of stone									
Single	72	90	26	86.67	10	50	108	83.08	0.014
Multiple	8	10	4	13.33	10	50	22	16.92	
Size of stone									
<1 cm	8	10	2	6.67	8	40	18	13.85	0.052
>1 cm	72	90	28	93.33	12	60	112	86.15	
Impacted stone at neck of GB									
Absent	80	100	26	86.67	10	50	116	89.23	0.001
Present	0	0	4	13.33	10	50	14	10.77	
Bile spillage									
Absent	80	100	20	66.67	2	10	102	78.46	0.001
Present	0	0	10	33.33	18	90	28	21.54	

Table 4: Association of grade of acute cholecystitis with CRP (N=130)

CRP (mg/l)	Mild cholecystitis (n=80)		Moderate cholecystitis (n=30)		Severe cholecystitis (n=20)		P-value
	n	%	n	%	n	%	
<1	80	100.00	0	0.00	0	0.00	0.001
1-6	0	0.00	20	66.67	0	0.00	
>6	0	0.00	10	33.33	20	100.00	
Mean±SD	0.646±0.231		5.300±1.76		13.78±4.48		

Table 5: Association of grade of acute cholecystitis with ESR (N=130)

Serum ESR (mm/hour)	Mild cholecystitis (n=80)		Moderate cholecystitis (n=30)		Severe cholecystitis (n=20)		P-value
	n	%	n	%	n	%	
14-18	70	87.50	0	0.00	0	0.00	0.001
19-38	10	12.50	28	93.33	0	0.00	
39-50	0	0.00	2	6.67	20	100.00	
Mean±SD	14.76±3.54		36.78±2.35		47.45±2.97		

Discussion

Acute cholecystitis is a common inflammatory condition of the gallbladder, often requiring timely diagnosis and severity assessment to guide treatment. Biomarkers like CRP and ESR have emerged as valuable tools for evaluating inflammation. This study aims to estimate the severity of acute cholecystitis using CRP and ESR levels, aiding in early risk stratification and management. The mean body mass index (BMI) was 25.7 ± 5.1 kg/m^2, which indicates that a significant portion of patients were overweight, a known risk factor for gallstone formation and subsequent cholecystitis. This aligns with findings from the CHOLE-COVID study, which reported a mean BMI of 27.1 ± 5.3 kg/m^2 among AC patients, underscoring the association between higher BMI and gallbladder disease[14]. In this study, the mean hemoglobin level was 12.5 ± 2.6 g/dL, The white blood cell (WBC) count averaged 11.8 ± 3.7 × 10^3/mL, indicative of a mild leukocytosis commonly observed in inflammatory conditions like AC. This is consistent with the study by Zgheib et al., which found that WBC counts were significantly higher in patients with moderate or gangrenous cholecystitis compared to those with milder forms[15]. Liver function tests revealed elevated levels of total bilirubin (1.56 ± 1.28 mg/dL), alanine aminotransferase (ALT) (50.6 ± 51.4 IU/L), and aspartate aminotransferase (AST) (55.3 ± 61.7 IU/L). These elevations may reflect biliary obstruction or hepatic inflammation associated with AC. Zgheib et al. reported similar findings, with mean total bilirubin levels of 1.82 mg/dL, ALT of 110.9 IU/L, and AST of 164.4 IU/L in patients with AC and concomitant common bile duct stones (CBDS), that highlights the impact of biliary obstruction on liver enzymes [15]. In our study of acute cholecystitis patients, most participants were middle-aged with the 41–50 age. Females slightly outnumbered males (55.38%). Although no significant association was found between gender and disease severity, older age was linked to more severe cholecystitis (p=0.02). These findings align with those of Gurbulak et al., who reported a female predominance and increasing severity with age (p<0.05)[16]. Similar age trends were noted by Muhammad et al. (mean age 40.32 years, 75% female)[17], Sakalar et al. (mean age 59.87 years, severity correlated with age but not gender)[18], and Park et al., who observed that patients with more severe disease were significantly older (p<0.05)[19]. The Tokyo Guidelines highlight key USG indicators of acute cholecystitis (AC), including probe tenderness in the area of gall bladder, GB wall thickness >4 mm, enlarged gall bladder, impacted gall stones, presence of debris or pericholecystic fluid collection and sonolucent layer in GB wall[20]. In our study, USG was performed in all cases. We observed GB wall thickening (>4 mm) in 9.23%, CBD dilation (>6 mm) in 15.38%, multiple stones in 16.92%, impacted stones at the GB neck

in 10.77%, and bile spillage in 21.54%. Most stones (86.15%) were >1 cm. Significant associations were found between AC severity and GB wall thickening, CBD dilation, multiple and impacted stones, and bile spillage ($p<0.05$), though stone size was not significantly associated ($p>0.05$). Supporting studies reported GB wall thickening in 47.3%, multiple stones in 81.1%, and CBD dilation in 20.3% of cases [21]. Indar et al. also found USG findings correlated with moderate AC severity ($p<0.05$) [22]. Another study noted increasing abnormal USG features with severity but without statistical significance [23]. According to the Tokyo Guidelines, CRP is used for diagnosing AC, but not for grading its severity. In our study, all severe cases had CRP >6 mg/L, while mild cases had CRP <1 mg/L. The mean CRP levels were 0.646 mg/L (mild), 5.30 mg/L (moderate), and 13.78 mg/L (severe), showing a significant correlation with disease severity ($p<0.05$). ESR, an acute-phase reactant, has limited clarity in diagnosing and grading AC. According to our study, higher ESR levels significantly associated with increased disease severity ($p<0.05$). ESR ranged from 14–18 mm/hr in 53.85% of cases, 19–38 mm/hr in 29.23%, and 39–50 mm/hr in 16.92%. Our results from CRP and ESR are comparable with the findings of Gurbulak et al [24]. Despite these findings, ESR is a non-specific marker influenced by many conditions, such as infections, autoimmune diseases, and pregnancy. Our results suggests that ESR increases with inflammatory burden, although its diagnostic accuracy is limited.

Limitations of the study:

- The cross-sectional design did not allow for evaluation of long-term outcomes such as complications, recurrence, or mortality.
- Although exclusion criteria were applied, other unmeasured comorbid conditions might have influenced CRP and ESR levels, potentially introducing bias.
- Severity classification relied on clinical and imaging findings without histopathological confirmation, which may lead to interobserver variability.

Conclusion

CRP and ESR levels show a clear and statistically significant correlation with the clinical severity of acute cholecystitis. CRP levels below 1 mg/L were consistently associated with mild disease, while values exceeding 6 mg/L strongly predicted severe inflammation. Similarly, higher ESR levels aligned with increasing severity grades. These markers, due to their accessibility and cost-effectiveness, can be valuable adjuncts to clinical and radiological assessment, especially in resource-limited settings. Future studies with larger sample sizes and multicenter participation are recommended to validate these cut-off values and further refine severity prediction tools for acute cholecystitis.

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Ethical approval: The study was approved by the Institutional Ethics Committee.

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Original Article

Oral Health Status of Pregnant Women: A Cross-Sectional Study in A Tertiary Level Hospital

Shameem SI[1]*, Ava KMM[1]

Abstract

Background: Pregnancy induces physiological changes that can increase the risk of oral diseases such as gingivitis, periodontitis, and dental caries. Despite the importance of oral health in maternal and fetal outcomes, oral health often receives low priority during prenatal care in many developing countries, including Bangladesh.

Objectives: This study aimed to assess the oral health status of pregnant women attending Khulna City Medical College Hospital and identify factors associated with poor oral health outcomes.

Methods: A cross-sectional study was conducted from January 2023 to January 2025, enrolling 200 pregnant women using systematic random sampling. A structured questionnaire and clinical oral examination (based on WHO oral health assessment form) were used. Data were analyzed using SPSS v26; associations were assessed using chi-square and logistic regression analyses.

Results: Of the 200 participants, 68% had gingivitis, 42% had dental caries, and 28% had periodontal pockets ≥4 mm. Poor oral hygiene (Simplified Oral Hygiene Index, OHI-S >3) was found in 61%. Low education level, low income, and irregular dental visits were significantly associated with poor oral health outcomes ($p < 0.05$). Incidence of gingivitis was higher in the 2nd and 3rd trimesters. Dental caries was more common in the 3rd trimester, possibly due to hormonal changes and dietary habits. The incidence of periodontal disease (pockets ≥4mm) was significantly higher in the 3rd trimester.

Conclusion: This study provides valuable insight into the oral health status of pregnant women at Khulna City Medical College Hospital. The high prevalence of gingivitis, dental caries, periodontal disease, and poor oral hygiene underscores the need for preventive care and oral health education. Pregnant women should be routinely screened for oral health conditions, and dental care should be integrated into prenatal care routines.

Keywords: Pregnancy, Pregnant Women, Oral Health, Gingivitis, Periodontitis, Dental Caries, Khulna, Bangladesh.

Ipex J 2025; 1(2) : 57-61

Introduction

Pregnancy is a period of profound physiological, hormonal, emotional, and behavioral changes that can significantly impact a woman's overall and oral health. Elevated levels of estrogen and progesterone during pregnancy enhance vascular permeability and increase the inflammatory response, particularly in the gingival tissues, making pregnant women more susceptible to gingivitis—a condition commonly referred to as "pregnancy gingivitis." These changes may begin as early as the first trimester and often worsen as pregnancy advances if appropriate oral hygiene is not maintained.

Additionally, pregnant women may experience a higher

1. Dr. Md. Shahidul Islam Shameem, BDS, FCPS (Oral & Maxillofacial Surgery), Assistant Professor and Head of the department, Department of Dental & Maxillofacial Surgery, Khulna City Medical College Hospital.
2. Dr. K M Marium Ava Mitumoni, BDS, MPH, Register, Oral & Dental Surgeon, Department of Dental & Maxillofacial Surgery, Khulna City Medical College Hospital.

Corresponding Author: Dr. Md. Shahidul Islam Shameem, Assistant Professor and Head of the department. Department of Dental & Maxillofacial Surgery, Khulna City Medical College Hospital.
E-mail: doctor.shameemoms@gmail.com

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incidence of dental caries, periodontal disease, tooth mobility, and oral erosions, often due to altered dietary patterns (such as increased carbohydrate intake or frequent snacking), nausea and vomiting associated with morning sickness, and reduced focus on maintaining oral hygiene. These oral health issues not only lead to discomfort, pain, and difficulty in eating but may also negatively affect maternal nutrition and overall quality of life during pregnancy.

Importantly, several studies have highlighted a strong association between periodontal disease and adverse pregnancy outcomes, such as preterm birth, low birth weight, and preeclampsia (Offenbacher et al., 1996; Xiong et al., 2006). The proposed biological mechanism involves the translocation of periodontal pathogens and pro-inflammatory cytokines into the systemic circulation, which may contribute to systemic inflammation and affect placental and fetal development.

Globally, the prevalence of oral diseases among pregnant women remains high. Studies from both high- and middle-income countries like the United States, Brazil, China, and India have reported gingivitis rates ranging from 60% to 75%, dental caries from 40% to 60%, and periodontal disease affecting 20% to 40% of pregnant women (George et al., 2011; Hashim et al., 2012).

However, oral health care during pregnancy continues to be undervalued in many countries, especially in low- and middle-income settings, where lack of awareness, insufficient training among antenatal care providers, cultural beliefs, and limited accessibility to dental services serve as major barriers to preventive and curative dental care.

In low- and middle-income countries, access to oral health care remains limited, and awareness among both pregnant women and health care providers is often low (Amin & ElSalhy et al., 2014).

In Bangladesh, oral health is not routinely integrated into antenatal care protocols, and national data on the oral health status of pregnant women is limited. Consequently, oral health problems during pregnancy are often unrecognized and untreated. Moreover, both pregnant women and healthcare providers often harbor misconceptions regarding the safety and necessity of dental treatment during pregnancy, leading to further neglect.

To address this gap, the present study was conducted to evaluate the oral health status of pregnant women attending Khulna City Medical College Hospital, a tertiary care facility in southern Bangladesh. The study aimed to evaluate the prevalence of oral conditions like gingivitis, dental caries, and periodontal disease, and to correlate these conditions with demographic factors such as age, trimester, and socioeconomic status.

The findings are expected to contribute to the existing body of knowledge and to advocate for the integration of oral health into routine maternal healthcare services.

Materials and Methods:

Study Design and Setting:
A hospital-based, cross-sectional study was conducted at Khulna City Medical College Hospital, Bangladesh, between January 2023 and January 2025.

Ethical Approval:
Approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants.

Sample Size:
A sample size of 200 pregnant women was calculated to ensure sufficient power.

Sampling Technique:
Systematic random sampling was used.

Inclusion Criteria:

- Pregnant women aged 18–40 years
- Willing to participate and provide consent
- Participants in their 1st, 2nd or 3rd trimester of pregnancy

Exclusion Criteria:

- Women with systemic diseases (e.g., diabetes)
- Women on medications affecting periodontal health (e.g., phenytoin)
- History of periodontal treatment in past 6 months
- Non-pregnant women

Data Collection Tools:

- Structured, interviewer-administered questionnaire (demographics, oral hygiene habits, dental visit history, diet)
- Clinical oral examination following WHO Oral Health Survey 2013 guidelines
- Gingival Index (GI): Was used to Assess gingival inflammation)

- Decayed, Missing, Filled Teeth (DMFT) Index: Was used to assess the presence of dental caries.
- Periodontal pocket depth assessment
- Simplified Oral Hygiene Index (OHI-S): Was used to assess oral hygiene level

Data Analysis:

Data were analyzed using SPSS v26. Descriptive statistics summarized prevalence rates. Chi-square tests assessed associations between categorical variables; logistic regression identified independent predictors. A p-value <0.05 was considered statistically significant.

Result and observation:

Demographic Characteristics of Participants:

The study included 200 pregnant women with a mean age of 27.6 ± 4.8 years. Most of the participants were in the 2nd trimester (45%), followed by the 3rd trimester (35%) and 1st trimester (20%). In terms of education level, the majority had secondary education (35%), followed by primary education (30%), and higher education (20%). The socioeconomic background showed that 40% of the participants had a monthly household income of <10,000 BDT.

Table 1. Participant Characteristics (n=200)

Variable	Category	Frequency	Percentage
Age (mean ± SD)	-	27.6 ± 4.8 years	(%)
Trimester	1st trimester	40	20%
	2nd trimester	90	45%
	3rd trimester	70	35%
Education Level	No formal education	30	15%
	Primary	60	30%
	Secondary	70	35%
	Higher	40	20%
Monthly Household	<10,000 BDT	80	40%
income	10,000 – 20,000 BDT	70	35%
	20,000 BDT	50	25%

Oral Health Findings:

Gingivitis was the most prevalent condition, affecting 68% of the participants. It was most common in the 2nd trimester. Dental caries (DMFT ≥1) was present in 42% of the participants, with the highest prevalence seen in the 3rd trimester. Periodontal pockets ≥4mm were found in 28% of the women, with more severe cases observed in those in the 3rd trimester. Poor oral hygiene (OHI-S >3) was reported in 61% of the pregnant women, indicating suboptimal oral hygiene practices.

Table 2. Oral Health Status among Pregnant Women (n = 200)

Oral Health Condition	Frequency	Percentage
Gingivitis 136	68%	(%)
Dental Caries (DMFT ≥ 1)	84	42%
Periodontal Pocket ≥ 4mm	56	28%
Poor Oral Hygiene (OHI-S > 3)	122	61%
Awareness of oral health	56	28%

Figure1. Prevalence of Oral Conditions among Pregnant Women

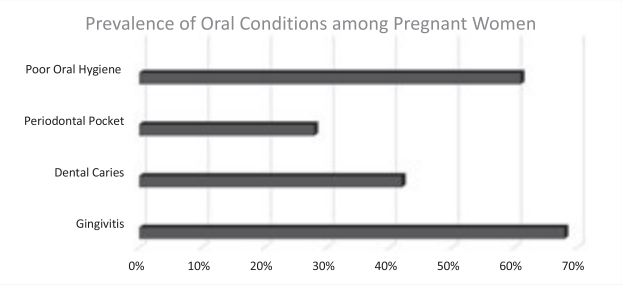


Figure 2: Awareness about pregnancy and oral health

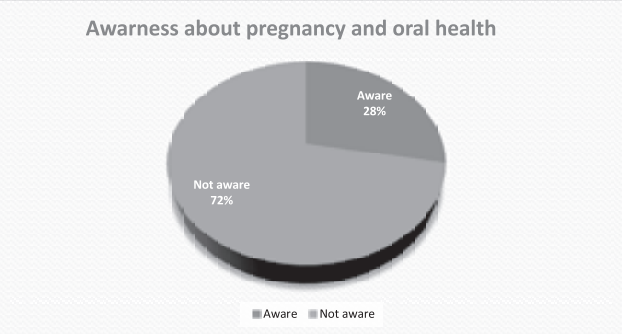


Table 3. Association of Socio-demographic Factors with Gingivitis

Factor	Gingivitis (%)	No Gingivitis (%)	P-value
Education: Low	80%	20%	0.01**
Education: High	50%	50%	
Income: Low	75%	25%	0.01**
Income: High	55%	45%	
Dental Visit: None	70%	30%	0.01**
Dental Visit: Regular	45%	55%	

Table 3 showed the relationship between various socio-demographic factors—such as education level, monthly income, and dental visits during pregnancy—and the presence of gingivitis among pregnant women in the study. The data show that lower education levels and lower household income are significantly associated with a higher prevalence of gingivitis ($p < 0.01$). Additionally, women who did not visit a dentist during pregnancy had a notably higher rate of gingivitis compared to those who did. These findings highlight the influence of socio-economic status and oral healthcare access on periodontal health during pregnancy.

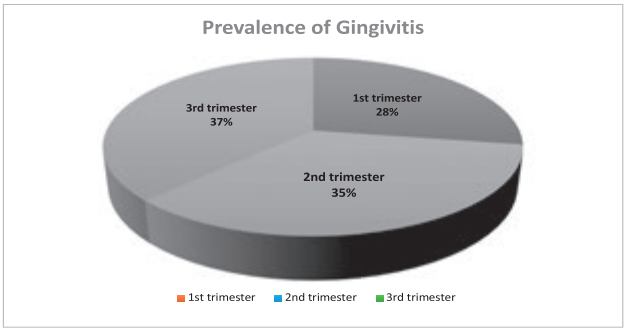
Trimester-wise Distribution of Oral Health Conditions:

Table 4. Oral Health Status in trimester

Oral Health Condition	1st trimester	2nd trimester	3rd trimester
Gingivitis	28%	35%	37%
Dental Caries	20%	33%	47%
Periodontal Pocket ≥ 4mm	28%	40%	42%

Table 4. showed the incidence of gingivitis was higher in the 2nd and 3rd trimesters. Dental caries was more common in the 3rd trimester, possibly due to hormonal changes and dietary habits. The incidence of periodontal disease (pockets ≥4mm) was significantly higher in the 3rd trimester.

Figure 2. Trimester-wise Gingivitis Prevalence



Discussion:

Oral health during pregnancy is a critical issue. Hormonal changes, increased blood flow, and alterations in the immune response can predispose pregnant women to conditions like gingivitis and periodontitis.

This study revealed a notably high prevalence of gingivitis (68%) among pregnant women attending Khulna City Medical College Hospital. This finding aligns with global studies reporting increased gingival inflammation during pregnancy due to hormonal surges. Elevated levels of estrogen and progesterone increase vascular permeability and modify the host immune response, predisposing gingival tissues to inflammation even in response to minimal plaque accumulation (Silk et al., 2008; George et al., 2011).

The trimester-wise analysis in our study showed an increase in gingivitis prevalence with advancing pregnancy, highest in the third trimester, corroborating the findings of Yenen et al. (2011), who noted progressive worsening of gingival conditions as pregnancy advances.

The prevalence of dental caries (42%) is also consistent with findings from other low-resource settings. Frequent snacking, increased intake of fermentable carbohydrates, morning sickness-induced vomiting, and reduced attention to oral hygiene are contributing factors (Hashim et al., 2012; Ahmed et al., 2020). Physiological changes in saliva, such as decreased flow and altered pH, further promote caries development during pregnancy.

Periodontal pockets ≥4mm were found in 28% of participants, indicating progression from gingivitis to periodontitis in a significant proportion. These findings are comparable to studies from the U.S. and China, where periodontitis prevalence among pregnant women ranged from 20–40% (Offenbacher et al., 1996; Xiong et al., 2006). The implications of maternal periodontitis are profound, as several studies have linked it to adverse pregnancy outcomes, including preterm birth and low birth weight (Bobetsis et al., 2006; Sanz et al., 2013).

Socio-demographic factors such as low education level, lower income, and poor oral hygiene habits were significantly associated with increased risk of gingivitis and caries in our study. Women with no formal education were 2.5 times more likely to develop gingivitis, emphasizing the role of health literacy in oral health maintenance. This is in line with findings from studies conducted in India and Jordan (Amin & ElSalhy, 2014; Al Habashneh et al., 2005), which also highlight the protective role of education and regular dental visits.

Alarmingly, only 15% of participants had visited a dentist during their pregnancy. This underutilization of dental services reflects a lack of awareness, socio-cultural misconceptions (e.g., the belief that dental treatment during pregnancy is harmful), and possibly limited availability of affordable care. Studies by Gaffield et al. (2001) and the CDC (Centers for Disease Control and Prevention) support these barriers, stressing the need for targeted oral health education during antenatal care.

Despite these risks, only 15% of participants had visited a dentist during pregnancy. This underscores the urgent need to integrate dental screening into routine antenatal care, as recommended by WHO.

This study showed that lower education levels and lower household income are significantly associated with a higher prevalence of gingivitis ($p < 0.01$). Additionally, women who did not visit a dentist during pregnancy had a notably higher rate of gingivitis compared to those who did. These findings highlight the influence of socio-economic status and oral healthcare access on periodontal health during pregnancy.

The findings from this study suggest a need for targeted oral health education for pregnant women, particularly in the second and third trimesters. It is essential to emphasize the importance of regular dental visits, proper brushing techniques, and the role of nutrition in maintaining good oral health during pregnancy.

Conclusion:

Oral health among pregnant women in Khulna is significantly compromised. Awareness programs and integration of oral health services into routine antenatal care are essential. Healthcare providers, including obstetricians and dentists, must collaborate to ensure comprehensive maternal care. The oral health status of pregnant women attending Khulna City Medical College Hospital was poor, with high rates of gingivitis, caries and periodontal disease. Addressing oral health in pregnancy is crucial for improving both maternal and fetal outcomes.

Recommendations:

- Implement community-based oral health education programs for pregnant women
- Provide affordable preventive dental services for pregnant women
- Encourage routine dental check-ups during prenatal visits
- Train healthcare professionals on the importance of maternal oral health
- Policy advocacy for inclusion of dental care in national maternal health programs

Limitations:

- Hospital-based sample may not represent the general pregnant population
- Self-reported data may have recall bias
- Cross-sectional design limits causal inferences

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Review Article

Premature Graying of Hair: Updates and Review

Nawaz UMS

Abstract

Background: Premature graying of hair (PGH), or premature canities, is a common condition characterized by the early loss of hair pigmentation. Despite being harmless, it frequently raises serious psychological and cosmetic issues, especially in young individuals.

Objective: This narrative review aims to provide a comprehensive overview of the current understanding of the etiology, pathogenesis, clinical associations, and management strategies of PGH.

Methods: A literature search was conducted to synthesize findings from clinical studies, review articles, and basic science research related to PGH, with emphasis on recent advances and emerging therapies.

Results: PGH is a multifactorial process which involves genetic predisposition, nutritional deficiencies, oxidative stress, autoimmune disorders, and lifestyle factors such as smoking and psychological stress. The primary mechanism involves the reduction or malfunction of melanocyte stem cells and follicular melanocytes. Despite the lack of a proven treatment, management strategies include lifestyle changes, cosmetic procedures, nutritional supplements, and experimental treatments including minoxidil, pseudocatalase, and melanocyte-stimulating drugs.

Advances in molecular biology and stem cell research may lead to targeted therapies in the future.

Conclusion: A complex interaction between biological and environmental factors is shown in premature graying of the hair. A deeper comprehension of melanocyte biology and oxidative stress creates new opportunities for possible therapies, even though the majority of current treatment is supportive. Further research is necessary to develop evidence-based, successful treatments.

Keywords: Premature graying, Canities, Hair pigmentation, Oxidative stress, Genetics, Melanocyte aging

Ipex J 2025; 1(2) : 62-69

Introduction

The commencement of hair depigmentation before the ages of 20 for Caucasians, 25 for Asians, and 30 for Africans is referred to as premature graying of hair, or early canities[1]. Premature graying is often regarded as a harmless cosmetic problem, yet it can have significant psychosocial repercussions, especially in younger people who may suffer from anxiety or social disengagement, or low self-esteem[2]. The condition reflects a complex interplay of genetic, environmental, nutritional, and oxidative factors, and may occasionally serve as an indicator of underlying systemic or autoimmune disease[3,4].

Growing aesthetic concerns and the need for efficient preventive or therapeutic measures have fueled interest in learning more about the pathophysiology of premature graying in recent years. Standardized management guidelines are missing, and the condition is still poorly understood despite its high frequency[3]. The purpose of this study is to review what is currently known about the causes, pathophysiology, related disorders, and existing therapies for premature graying of the hair.

Dr. UM. Shah Nawaz, Graded specialist in Dermatology and Venerology, CMH Saidpur

Corresponding Author: Dr. UM. Shah Nawaz, Graded specialist in Dermatology and Venerology, CMH Saidpur, Email- nawazshsmc05@gmail.com

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This review aims to explore the current knowledge regarding the etiology, pathophysiology, associated conditions, and available treatment approaches for premature graying of hair. Additionally, recent advances in molecular research and potential future directions will be discussed.

Definition and Epidemiology-

Premature hair graying or premature canities is a depigmentation phenomenon of hair before 20 years in Caucasians, before 25 years in Asians, and before 30 years in Africans[5]. This definition is based on ethnic and racial differences in melanin biology and hair pigmentation patterns. The condition is usually idiopathic, but on occasion, it could be a presentation of underlying systemic or nutritional deficiencies, more so in young individuals[6].

The prevalence of premature graying varies geographically and racially. One study of 15–25-year-old Indian students reported an incidence of around 25%[7], but a second study in Korean adolescents provided a lower figure, with the potential for environmental and genetic factors affecting susceptibility[8]. Males may be potentially more affected than females, although evidence has been inconsistent in studies[9].

Premature graying commonly begins in the frontal scalp and temples before progressing to the vertex and occipital scalp. Onset is either gradual or abrupt and initially tends to manifest in the scalp hair, but also other hairs of the body (beard, eyebrows, chest) can be involved[10]. While the condition itself is normally not dangerous, early onset has a profound influence on individuals' self-esteem, resulting in cosmetic discontent and psychological morbidity in patients who are affected[11].

Pathophysiology of Hair Pigmentation-

The hair pigmentation is determined by the presence, amount, and structure of melanin synthesized by the melanocytes in the hair follicle bulb. The melanocytes are of neural crest origin and migrate to the hair follicle during embryogenesis. Melanocytes play a role in melanin deposition on to keratinocytes of the hair shaft during anagen (growth) phase of the hair cycle[12,13].

The two primary types of melanin are eumelanin (brown-black pigment) and pheomelanin (yellow-red pigment). The relative proportion determines the vast variety of hair color in humans[14,15]. Pigmentation of the hair is tightly regulated by several signaling pathways, such as stem cell factor (SCF)/c-Kit, microphthalmia-associated transcription factor (MITF), and endothelin-3[16].

Hair graying, or canities, is caused by a progressive and cumulative reduction in pigment-forming melanocytes in the hair bulb. Oxidative stress, an imbalance between ROS and antioxidant defense systems, is one such underlying cause. Hydrogen peroxide (H_2O_2), a ROS that accumulates in senescent hair follicles, has been shown to inactivate the key melanogenic enzyme tyrosinase, thereby impairing melanin formation[17].

Also, with progressive aging and oxidative damage, there is loss of melanocyte stem cells that are found in the bulge region of the hair follicle. Inability to regenerate differentiated melanocytes in the hair bulb results in irreversibility of depigmentation[18]. Reduced activity of antioxidant enzymes such as catalase and glutathione peroxidase also perpetuates ROS in the follicular environment[19] .

Mitochondrial dysfunction, DNA damage, and faulty repair mechanisms have also been attributed to melanocyte senescence and apoptosis. These cellular aging mechanisms combined with the silencing of melanin production and the formation of gray or white hair[20].

Etiological Factors of Premature Graying

Premature graying is a multifactorial condition. A range of genetic, nutritional, environmental, and systemic factors have been implicated in its pathogenesis.

a. Genetic Predisposition

Genetics is one of the most well-established contributors to premature graying. An associated family history can be found in a majority of cases, suggesting strong genetic influences. Twin studies have supported the role of inherited factors in hair pigmentation and graying patterns[21].

Genetic loci such as IRF4, which regulates tyrosinase gene expression, and BCL2, which regulates survival of the melanocytes, have been linked to the timing of graying[22,23]. Polymorphisms in MC1R and TYR genes also modulate melanin production and pigmentation pathways.

b. Nutritional Deficiencies

Several micronutrient deficiencies have been associated with premature graying. These include:

- Vitamin B12: Essential for the production of red blood cells and DNA. Premature graying is significantly linked to its lack[24].
- Iron: Important for the activity of tyrosinase, an enzyme in melanin synthesis[24,25].
- Copper: Required as a cofactor for tyrosinase. Low levels impair melanin formation[25].
- Zinc and Calcium: Essential for hair follicle health and pigmentation regulation[26].

Studies in young adults have consistently shown that low serum levels of these elements are more frequent in individuals with premature graying.

c. Oxidative Stress

Oxidative stress is a central mechanism in the pathogenesis of premature graying. Reactive oxygen species (ROS), such as hydrogen peroxide, accumulate in hair follicles and damage melanocytes[26]. ROS are typically neutralized by antioxidant enzymes such as glutathione peroxidase and catalase. In individuals with premature graying, these enzymes are often found to be reduced[27].

The oxidative theory is further supported by the observation that melanocyte stem cell depletion and apoptosis occur in response to oxidative DNA damage[28].

d. Psychological and Physical Stress

The link between psychological stress and premature graying is becoming more widely recognized. Elevated levels of cortisol and other stress hormones may disrupt melanocyte homeostasis and lead to early apoptosis[29].

A landmark study demonstrated that sympathetic nervous system activation under stress depletes melanocyte stem cells in mice, providing experimental evidence for this mechanism[30].

e. Autoimmune and Endocrine Disorders

Localized or diffuse depigmentation can be caused by the loss or malfunction of melanocytes in autoimmune illnesses such vitiligo, alopecia areata, and pernicious anemia[31].

Premature graying is also frequently linked to thyroid conditions, particularly hypothyroidism. Although the precise process is unclear, it might have to do with altered stem cell activity or melanin synthesis[31].

f. Lifestyle and Environmental Factors

Several environmental and lifestyle elements have been implicated:

- Smoking: Significantly associated with early graying. Smokers have a 2.5 times greater risk than non-smokers[32].
- Pollution and UV radiation: May induce oxidative stress in hair follicles.
- Sedentary lifestyle and irregular diet: May contribute indirectly via nutrient deficiencies and metabolic stress[33].

Associated Conditions

despite the fact that it can occasionally be linked to inherited or systemic disorders, premature graying of the hair can also be a standalone cosmetic concern. To rule out underlying illnesses and comprehend the wider ramifications of early canities, it is critical to identify these relationships.

1. Genetic Syndromes Associated with Premature Graying

Certain premature aging disorders and syndromes feature graying of hair as one of their early signs. These include:

- Werner Syndrome: Often referred to as adult progeria, this autosomal recessive condition is typified by early graying, skin atrophy, cataracts, and premature aging[34].
- Progeria (Hutchinson-Gilford Syndrome): A uncommon genetic disorder that causes children to age quickly, frequently accompanied by graying and hair loss[35] .
- Rothmund-Thomson Syndrome: Involves skin atrophy, photosensitivity, short stature, and early hair graying[36].

2. Autoimmune Disorders

Several autoimmune diseases affect melanocytes or interfere with hair follicle functioning:

- Vitiligo: Characterized by loss of melanocytes, which can also affect hair follicles leading to depigmented hair[37].
- Alopecia Areata: Sometimes associated with regrowth of depigmented (white) hairs after hair loss, suggesting melanocyte targeting[38].
- Autoimmune thyroid disorders (particularly hypothyroidism) are frequently associated with early hair graying. The hypothyroid state may impact melanin production and melanocyte stem cell maintenance[39].

3. Other Systemic Associations

- Pernicious Anemia: a disorder brought on by a lack of vitamin B12 as a result of autoimmune intrinsic factor degradation. Premature canities and anemia may result from this[40].
- Atopic Diathesis: Atopic people, particularly those with atopic dermatitis, have been observed in certain studies to exhibit early graying, which may be related to immunologic pathways or oxidative stress[39].

4. Dermatologic Associations

- Tuberous Sclerosis Complex (TSC): May feature areas of white or gray hair in patches as part of its cutaneous manifestations[41].
- Neurofibromatosis: Café-au-lait spots and localized hair graying over neurofibromas may be noted[42].

Investigations

A common benign and idiopathic condition is premature graying of the hair (PGH), especially in those with a strong family history. Investigations, however, can be necessary in some clinical situations, particularly when systemic symptoms are present if the patient is young or has no family history. Eliminating underlying dietary, endocrine, autoimmune, or hereditary factors is the aim.[39,42]

1. When to Investigate

It is not usually required to undergo routine laboratory tests, especially if PGH manifests alone without systemic symptoms. Investigations are recommended, nevertheless, when:

- Onset is before age 20 without a family history[43]
- Accompanied by signs of nutritional deficiencies (e.g., fatigue, pallor)
- Associated with autoimmune symptoms (e.g., vitiligo, alopecia areata)
- Signs suggestive of thyroid dysfunction
- Suspicion of genetic syndromes (e.g., short stature, cataracts, skin atrophy)

2. Recommended Baseline Investigations

Test	Purpose	Indications
Complete Blood Count (CBC)	Detect anemia, especially megaloblastic	Fatigue, pallor, poor diet
Serum Vitamin B12	Check for B12 deficiency	Common in PGH, especially vegetarians[24]
Serum Ferritin and Iron Studies	Evaluate iron status	Important for melanin synthesis[44]
Serum Copper and Zinc	Check trace element status	Linked to tyrosinase activity[45]
Thyroid Function Tests (TSH, FT4)	Detect thyroid disorders	Hypothyroidism is a known PGH cause[46]
ANA (Antinuclear Antibody)	Screen for autoimmune diseases	If other signs of autoimmunity present
Lipid Profile and Fasting Glucose	Optional	If metabolic syndrome suspected in young patients

3. Special Tests (If Clinically Indicated)

- Skin biopsy: Rarely needed; considered only if diagnostic uncertainty or suspicion of another hair/scalp disorder.
- Genetic testing: Only in suspected progeroid syndromes or other inherited disorders.
- Serum homocysteine & methylmalonic acid: For borderline B12 deficiency confirmation.

4. Imaging and Hormonal Tests

Usually not necessary until systemic symptoms are in question. However, patients exhibiting symptoms of pituitary or adrenal dysfunction may be evaluated for hormonal assays (e.g., serum cortisol, testosterone, DHEAS).

Management Approaches

There isn't yet a treatment for premature graying of hair (PGH) that works for everyone. The management is primarily supportive, concentrating on providing lifestyle and cosmetic remedies as well as addressing underlying problems where found. Future treatment approaches may be provided by ongoing studies into the biology of hair pigmentation.

1. Nutritional Supplementation

Correction of micronutrient deficiencies can be helpful in selected patients, especially when lab investigations reveal low levels.

- Vitamin B12: Supplementation may reverse graying in some cases related to deficiency[47].
- Iron, Copper, Zinc, Calcium: These are essential cofactors for melanin synthesis; supplements may slow or reverse PGH in deficient individuals[39].
- Biotin, Folic Acid, and Vitamin D: Often included in over-the-counter hair supplements, though evidence is limited[24].

Note: Unless there is a compelling clinical suspicion, supplements should be targeted and evidence-based rather than empirical.

2. Topical and Systemic Agents

A. Cosmetic Options

- Hair Dyes: The most commonly used approach.
- Permanent dyes: Offer long-lasting results but may contain harsh chemicals (e.g., ammonia, PPD).
- Natural dyes: Henna, indigo – safer but may not offer uniform coverage.

B. Investigational Treatments

Several experimental therapies have shown promise in slowing or reversing graying, although most are not yet standard of care:

- Topical Melitane™ (a biomimetic peptide): Acts as an α-MSH agonist to stimulate melanogenesis[48].
- Pseudocatalase with calcium and UVB: Proposed for repigmentation in vitiligo and PGH, although data is limited[49].
- Topical minoxidil: May indirectly promote melanocyte function, but not consistently effective in PGH [50].
- Melatonin or topical melatonin analogs: Antioxidant effects are being explored in studies.

These treatments are not FDA-approved for PGH and should be considered experimental.

3. Lifestyle Modifications

Changes in lifestyle may help slow the progression of PGH:

- Smoking cessation: Strongly recommended as smoking is a well-established risk factor[51].
- Stress management: Yoga, mindfulness, and cognitive behavioral therapy may help reduce oxidative stress pathways[52].
- Balanced, antioxidant-rich diet: Includes fruits, vegetables, nuts, seeds, and lean protein sources.
- Adequate sleep and hydration: Crucial for maintaining hormonal balance and oxidative control.

4. Psychological Support

- For many individuals, PGH causes psychosocial distress. Counseling may be helpful for self-image issues, especially in adolescents or young adults.
- Support groups or online communities may also be beneficial.

5. Emerging and Future Therapies

- Stem cell therapy: Research on melanocyte stem cell transplantation is ongoing.
- Gene editing (CRISPR): Might target genes like IRF4 or Bcl2, implicated in hair pigmentation.
- Antioxidant therapies: New delivery systems (e.g., liposomes, nanocarriers) are being tested.

These remain in the experimental or early research phase, but show promise for future treatment modalities.

Recent Advances and Future Perspectives

Premature graying of hair (PGH) pathophysiology has become better understood thanks to recent developments in molecular biology, genetics, and stem cell research. In the near future, these findings could lead to new diagnostic instruments and focused treatment choices.

1. Melanocyte Stem Cell Research

Hair pigmentation depends on the renewal and migration of melanocyte stem cells (McSCs) located in the bulge region of the hair follicle. Loss, exhaustion, or incorrect migration of these stem cells is believed to be a primary cause of irreversible graying[53].

- Animal studies (e.g., in mice) have shown that oxidative stress and aging cause melanocyte stem cells to differentiate prematurely, depleting the stem cell reservoir[54].
- Therapeutic targeting of stem cell niches may offer potential for reversing or delaying graying in the future.

2. Genetic Discoveries

Advancements in genome-wide association studies (GWAS) have identified key genes involved in hair pigmentation:

- IRF4: Associated with regulation of melanin production and linked with premature graying in European populations[55].
- BCL2 and MITF: Involved in melanocyte survival and melanogenesis.
- TYR and TYRP1: Enzymes directly related to melanin synthesis.

These findings may pave the way for gene-targeted therapies or predictive genetic testing in high-risk individuals.

3. Oxidative Stress Pathway Targeting

Research is focusing on mitigating oxidative damage in the hair follicle environment:

- Antioxidant-based topicals (e.g., catalase mimetics, melatonin analogs) are under investigation.
- Use of nanotechnology and targeted delivery systems to transport antioxidants directly to hair follicles is a promising area[27].

4. Role of Microbiome and Epigenetics

According to this research, the start of PGH may be influenced by the scalp microbiota and epigenetic changes (such as variations in DNA methylation). Though mostly untapped, these sectors have the potential for innovative treatments.[56]

5. Artificial Pigmentation & Cosmetic Biotechnology

- Advances in safe, long-lasting hair dyes using plant-based or enzymatic colorants.
- Biotech companies are exploring bio-identical melanins for cosmetic repigmentation.

6. Ongoing Clinical Trials

Although there aren't any FDA-approved medications to reverse PGH at the moment, ongoing study is looking at substances that might prevent graying, such as:

- Topical melanin-stimulating peptides
- Stem cell-activating lotions
- Oral nutraceuticals with antioxidant and trace element combinations

The future of PGH management is likely to involve multimodal approaches—combining genetics, nutrition, topical therapy, and stem cell biology.

Conclusion

Premature graying of hair (PGH) is a multifactorial condition influenced by genetic, nutritional, environmental, and systemic factors.. Despite being mostly a cosmetic issue, in some individuals it could be an indication of underlying systemic disorders or nutritional deficiencies.

With a focus on oxidative stress, melanocyte stem cell depletion, and genetic regulation of melanin formation, our understanding of the pathophysiology has undergone tremendous change. Even though the majority of present management techniques are supportive or cosmetic, there is optimism for future therapeutic interventions due to ongoing research.

Current clinical practice continues to be based on timely diagnosis of reversible causes, individualized management approaches, and patient counseling. Effective preventive or restorative treatments may eventually result from ongoing research into melanocyte biology and molecular processes.

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Case Report

Endoscopic Assisted Evacuation of Chronic Subdural Hematoma

Ahmed MF

Abstract

Background: Chronic subdural hematoma (CSDH) is a common neurosurgical condition, typically managed with burr-hole evacuation. Although widely used, this method carries a higher recurrence risk compared to craniotomy. Endoscopic-assisted evacuation has recently emerged as a minimally invasive technique that provides better visualization and may reduce recurrence.

Case Presentation: We report a 72-year-old Bangladeshi male with a history of head trauma 20 days prior, presenting with altered consciousness and right-sided hemiparesis for 10 days. He was a known diabetic, hypertensive, and post-CABG patient with left ventricular failure. On admission, his Glasgow Coma Scale (GCS) was 7/15 with anisocoria. CT scan revealed a chronic subdural hematoma with an estimated volume of 50 ml.The patient underwent left parietal burr-hole evacuation under endoscopic guidance using the YKD-9101 system. Pseudo-membrane disruption and clot evacuation were achieved, followed by subdural drain placement. Postoperative CT showed significant reduction of hematoma volume (50 ml 15 ml). Clinically, GCS improved from 7/15 to 13/15, and right-sided muscle power improved from 2/5 to 4/5.

Conclusion: Endoscopic-assisted burr-hole evacuation provides superior visualization of hematoma membranes and cortical vessels, facilitating effective evacuation and potentially lowering recurrence risk. This case highlights its safety and efficacy in the management of CSDH in elderly patients with multiple comorbidities.

Keywords: Chronic subdural hematoma, Endoscopic evacuation, Burr-hole surgery

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Introduction

Chronic subdural hematoma is a common surgically treated intracranial emergency. Chronic subdural hematomas (CSDH) are characterized by the formation of a blood envelope between the dura mater and arachnoid mater (subdural space). It is a benign, slow-growing intracranial hemorrhage that contains a mixture of fluid and clotted blood.1 A projection made with original and reviewed data suggests that the incidence may increase by 53 % by the year 20403. One of the main reasons for this increase is populational aging, as age is the most important risk factor for CSDH.3 The most common history findings were history of trauma (69.9 %), motor deficit (68.4 %) and cognitive deficit (26.3 %). The average hematoma size was similar on both sides, and showed an increasing trend with aging. The size of CSDH was also greater in those who presented motor deficits.

Compression of the brain parenchyma and intracranial hypertension can be caused by incremental hematoma, which may result in clinical symptoms such as motor disturbance, gait abnormality, headache, cognitive disturbance, and aphasia. The first choice of management in these patients is a large craniotomy or burr-hole evacuation.

Dr. Md. Farhad Ahmed, Assistant Professor, Department of Neurosurgery, National Institute of Neurosciences & Hospital

Corresponding Author:
Dr. Md. Farhad Ahmed, Assistant Professor, Department of Neurosurgery, National Institute of Neurosciences & Hospital
Email: farahm1803@gmail.com

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Burr-hole evacuation is the most widely used technique, as it is both minimally invasive and relatively quick1. However, the recurrence rate of burr-hole evacuation is slightly higher than that of osteoplastic craniotomy due to limited exposure or involuntary injury[1].

Recently, endoscopic evacuation has been reported to a be a feasible method for evacuation in acute, subacute and chronic SDH patients in different age groups with favourable outcome 1–3. It is also reported that hematoma clots, trabecular structures, and stretching of the cortical vessels, which could be found only under neuro-endoscopy, were significant risk factors for CSDH recurrence[1].

Here we report our experience on a typical case on chronic subdural hematoma and its evacuation with the assistance of endoscope.

Case presentation:

A 72-year-old Bangladeshi male, hailing from Meherpur, presented in MICARE health with complaints of history of accidental fall with head trauma 20 days back, altered consciousness for 10 days and right sided hemiparesis for same duration.

The patient is diabetic, hypertensive and a diagnosed case of LVF. He had a history of undergone CABG surgery in 2016 with no significant post-operative complications. On physical examination, his BP- 160/110 mm Hg, Pulse 98 bpm, SPO2 98%, Temperature normal. Clinical examination revealed GCS E2M4V1 (7/15) and anisocoria with a sluggishly reacting left pupil. After completing examinations and investigations, decision was taken for operative measures for definitive treatment for the patient.

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Per operative findings:

A Parietal burr hole made on Left side on parietal eminence. Burr hole diameter 2 cm for easy maneuverability of endoscope. "YKD-9101 Full Endoscope System" with 3 mm Telescope used for this surgery. 'X'-shaped durotomy done, cauterized for widening Dural window. Then endoscope inserted keeping the telescope at air media. An angled 2 mm zero sucker used for suction-evacuation of Subdural haemorrhage. Sucker also used for disrupting pseudo membrane of Subdural Haematoma. Pseudo membrane disruption helped trapped haemorrhage to come out easily. (Fig: 1) After evacuation Brain pulsation seen. A Subdural drain inserted under Endoscopic guidance. A haemostatic Gel foam kept on burr hole site beside the drain tube. Skin closed in single layer. Drain tube removed on 3rd post-operative day

To objectively assess the patient's condition before and after surgery, several clinical parameters were recorded (Table: 1)

Parameter	Preoperative	Postoperative
CT Scan Volume of Hematoma	50 ml	15 ml
Glasgow Coma Scale (GCS)	7/15	13/15
Muscle Power (Right Side)	2/5	4/5

Table 1: Clinical parameters before and after evacuation of Chronic subdural hematoma

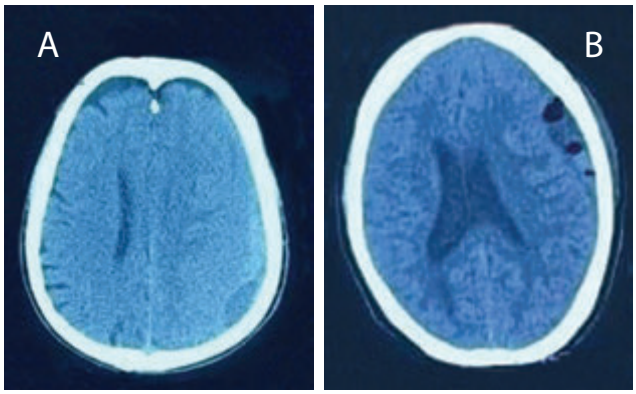

Fig: Preoperative (A) and post-operative (B) CT scan image

Discussion

Chronic subdural hematoma (CSDH) is a common neurosurgical condition, particularly prevalent among the elderly population. The gradual accumulation of blood in the subdural space, often following minor or unrecognized trauma, can result in significant neurological deficits due to cortical compression and increased intracranial pressure. Age-related cerebral atrophy, anticoagulant use, and comorbidities such as hypertension and diabetes are recognized predisposing factors[2,3].

In this case, the patient was a 72-year-old male with a history of hypertension, diabetes mellitus, and previous coronary artery bypass grafting (CABG). He presented with neurological deficits following a fall, and radiological imaging revealed a sizable chronic subdural hematoma. The patient underwent endoscopic-assisted evacuation using neuroendoscopes, which enabled complete visualization and removal of the hematoma components.

Traditionally, burr-hole drainage has been the most commonly employed surgical approach for CSDH due to its minimally invasive nature and relative simplicity. However, recurrence rates following burr-hole procedures remain a concern, with contributing factors including residual hematoma clots, septations, and insufficient cortical expansion post-surgery[1,3]. Craniotomy offers better visualization but is more invasive and not always ideal for elderly or comorbid patients.

Endoscopic evacuation presents a compelling alternative, as it allows direct visualization of the subdural space, enabling the identification and removal of septations, organized clots, and stretching of cortical vessels—features that are often missed during conventional surgery[1]. In the current case, postoperative improvement was significant: hematoma volume reduced from 50 ml to 15 ml, Glasgow Coma Scale (GCS) improved from 7/15 to 13/15, and motor power on the affected side increased from 2/5 to 4/5.

Our findings are consistent with those of Mokbul et al., who demonstrated the efficacy of both rigid and flexible endoscopic evacuation of CSDH in preventing recurrence and promoting faster neurological recovery[1]. Similarly, Zhu et al. highlighted that detailed intraoperative visualization and complete removal of hematoma membranes and clots are key factors associated with lower recurrence rates [3]. Additionally, Karam et al. emphasized the growing incidence of CSDH with age and the need for optimized surgical strategies tailored to geriatric patients[2].

The endoscopic technique also offers other advantages: smaller craniotomy size, reduced operative time, and suitability under local or general anesthesia—all beneficial for high-risk patients[1]. While the availability of neuroendoscopic expertise and equipment may limit its widespread use, its role is expected to expand as surgical experience and technology improve.

This case underscores the potential benefits of endoscopic-assisted evacuation in managing chronic subdural hematomas, particularly in older adults with comorbidities. Further comparative studies and long-term follow-up data will be essential to establish standardized guidelines for endoscopic management in CSDH.

References

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